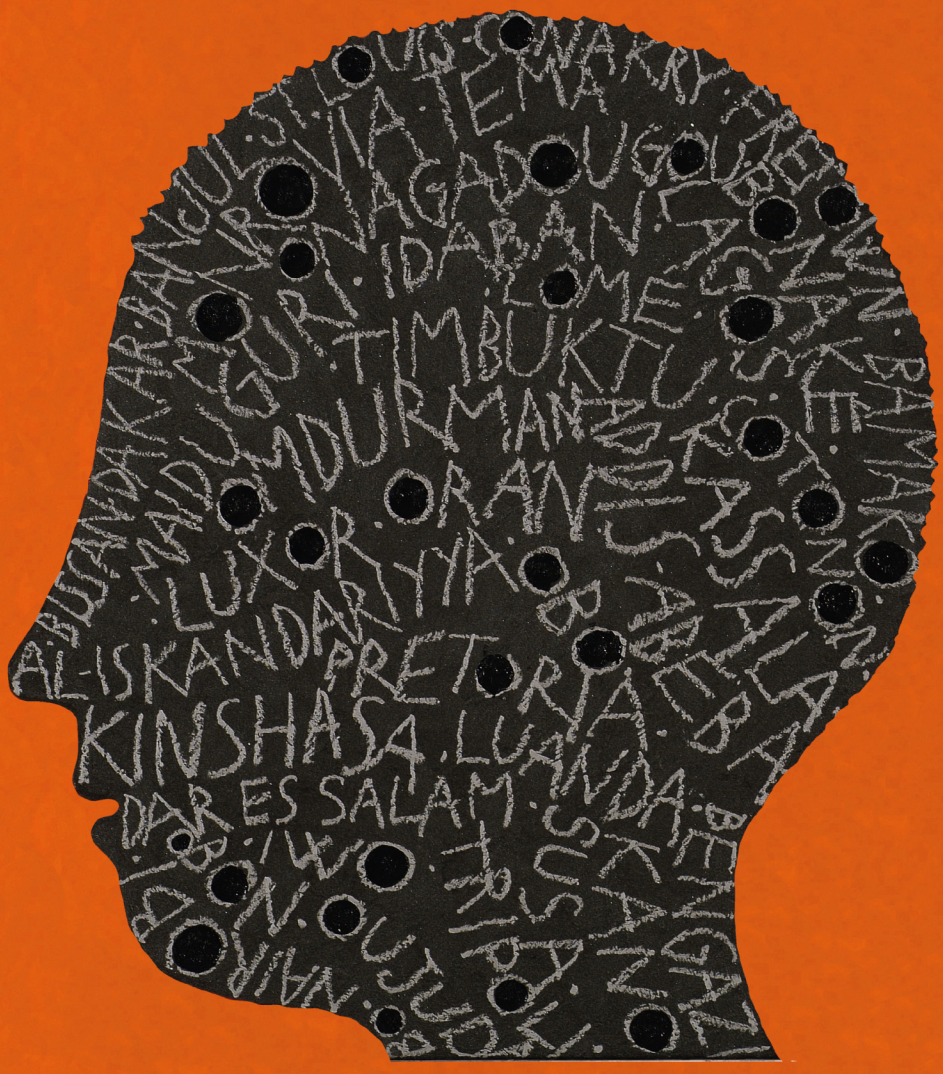
Volker Roder · Daniel R. Müller
Hans D. Brenner · William D. Spaulding

Integrated Psychological Therapy (IPT) for the Treatment of Neurocognition, Social Cognition, and Social Competency in **Schizophrenia** Patients



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Foreword

It is a special privilege to be asked to write a foreword for this book, which is the fruit of over two decades of visionary and ground-breaking work in support of the recovery process in schizophrenia and, indeed, other psychotic disorders. I recall visiting Bern in the late 1980s, as a young academic psychiatrist, when this work was being formulated and pioneered, and feeling very excited that a scientific approach to the psychosocial care of patients was in development. Bern was one of the very few places in the world providing leadership in this field. It attracted a cohort of like-minded people and inspired many of them to pursue this type of approach in their own settings. At the time there were very few therapeutic tools in the toolkit to treat psychotic patients apart from basic nursing care and antipsychotic medications. We are now in a much better position, although still far too few patients around the world are given access to truly holistic recovery-oriented care from the onset of their illnesses. If this were to occur, the burden of these illnesses would be dramatically lessened.

Based originally on stress/vulnerability and cognitive neuroscience models, IPT has covered all of the domains that are affected in schizophrenia from neurocognition and social cognition through relationships and social functioning in the real world. It has been evolved and evaluated progressively over a long period of time and now has an impressive evidence base to support its more widespread dissemination. This comprehensive text is a vital tool in this endeavor. People with schizophrenia or other psychoses and their families have many reasons to be grateful to these pioneers, committed therapists, and researchers who have produced this body of expertise. This new volume provides a highly accessible resource for clinicians and I really hope IPT is taken up much more widely within clinical care settings across the world so that many more patients and families can benefit.

Patrick McGorry, MD, PhD
University of Melbourne, Australia

Preface

The importance of cognitive factors in the therapy and rehabilitation of schizophrenia patients has been recognized, examined, and is increasingly being accepted internationally in recent years. Cognitive factors have proved to have a decisive influence on how successfully patients can be (re)integrated in the community ("recovery perspective").

The initial development of Integrated Psychological Therapy (IPT) in the 1980s was truly pioneering, since it was one of the very first comprehensive and manualized treatment approaches for schizophrenia patients that combined neurocognitive, social cognitive, and social competence interventions. It was also truly innovative, in that – for example – it already contained interventions addressing "social cognition" years before that term had even been defined. The original IPT manual, published in 1988 in German, became a sort of therapy "classic." Subsequently, both the theoretical background and practical procedures used in IPT were continuously adapted within this framework to reflect the latest results of clinical and basic research. The ultimate goal of IPT is to provide our patients with the best possible chances of a good recovery.

A total of 35 studies on IPT, involving 1,529 patients from 12 different countries in Europe, North, Middle, and South America, as well as Asia, have now been published (see Chapter 7). IPT is, therefore, one of the most widely studied approaches around, and a broad range of clinical experience and empirical results have been gathered over the years. The American Psychological Association (APA) has also adopted IPT in its recommendations for treating schizophrenia patients ("Catalog of Clinical Training Opportunities: Best Practices for Recovery and Improved Outcomes for People with Serious Mental Illness": http://www.apa.org/practice/resources/grid/index.aspx; June 2010).

The IPT manual itself has been published in 13 languages and in multiple editions. The first manual appeared more than 20 years ago, and the latest version (the 6th revised German edition) was published in 2008. Because the first (and to date only) English edition was last published in 1994 – and our knowledge and understanding of schizophrenia have improved immensely since then – we felt it was time to publish a new version. We, therefore, completely rewrote most of the chapters for this book and expanded it greatly.

The book is divided into three main parts:

– **Part A: Theoretical Background and Treatment Approaches: An Overview** contains two chapters. Chapter 1 describes the theoretical background of cognitive behavioral treatments, for example, vulnerability-stress models, and shows how neurocognition and social cognition are highly relevant variables for functional out-

come and recovery. Chapter 2 provides an overview of the different cognitive-behavioral treatment approaches (cognitive remediation therapy and cognitive therapy, social competence approaches, psychoeducation and family therapy, and integrative approaches) as well as empirical evidence concerning their efficacy.

– **Part B: IPT – Indication, Therapy, Assessment, and Evaluation,** comprises five chapters and forms the core of the book. Here, clinicians learn step by step how to use the therapy techniques of the five IPT subprograms and how to select the appropriate therapy materials (which themselves are described at the back of the book). Numerous vignettes and examples from real therapy sessions are used to illustrate the techniques. We also provide practical advice for dealing with (difficult) group processes as well as individual dyadic situations. Furthermore, therapists can learn how to use IPT within multimodal therapy and rehabilitation efforts (case management). Chapter 3 describes the conditions needed to carry out the therapy program, while Chapter 4 gives an overview of the IPT approach and its five subprograms. Chapter 5 then shows how these five IPT subprograms can be implemented in practice. Chapter 6 looks at assessment and treatment planning. Finally, Chapter 7 discusses empirical results of studies involving IPT.
– **Part C: Further Development of IPT.** The cognitive part of IPT is now directly oriented toward the NIMH MATRICS variables (Measurement and Treatment Research to Improve Cognition in Schizophrenia) for patients who are better socially integrated and show fewer negative symptoms. The social competence part has been expanded in three specific areas: residential, vocational, and recreational rehabilitation. New therapy programs were developed and evaluated for these areas.

Last, but not least, we would like to thank all patients as well as numerous clinicians and researchers for their (critical) feedback about IPT. This feedback has contributed enormously to the continuous development of IPT and to making it what it is today: a proven, empirically supported, and practical approach to treating schizophrenia. Our thanks also go to Anna Heuberger, MSc, and Manuela Christen, MSc, research psychologists at the Psychiatric University Hospital in Bern, Switzerland, who provided us with tremendous support in drafting and writing the different chapters of this book. Finally, we would also like to thank Hogrefe Publishing, especially Robert Dimbleby, who initiated this new edition of the IPT.

Bern, Switzerland, Summer 2010
Volker Roder

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B. IPT: Indication, Therapy, Assessment, and Evaluation

* We use the German abbreviation for "**W**ohnen, **A**rbeit, **F**reizeit."

Part A
Theoretical Background and Treatment Approaches: An Overview

1 Theoretical Basis of Cognitive Behavioral Treatments

1.1 Systemic Vulnerability – Stress Models

Theoretical models for understanding schizophrenia-spectrum disorders have evolved over many decades. By the end of the 1980s, the most widely accepted models included multiple causal or etiological factors. Schizophrenia is not attributed to any sole origin, but to the contributions of multiple biological, psychological, and social factors over the course of development. Similarly, these multiple contributions do not occur in simple, unidirectional ways; linear etiological models, in which causal processes cascade from a single origin to produce the disease, have been replaced by systemic models, in which causal processes interact with each other in circular, reciprocal ways.

Vulnerabilities are abnormalities or impairments in specific systemic processes. They do not directly cause the disease, but rather interact with other vulnerabilities and environmental challenges, or stress, to produce the disease state. Research on vulnerability to schizophrenia has focused on genetic factors and their endophenotypes (e.g., anatomical abnormalities, abnormal distribution of neurotransmitter receptors, neurocognitive impairments), although vulnerabilities could also be acquired, for example, deficits in self-regulation skills. Because of the dual role of enduring vulnerabilities and environmental factors, these models are generally known as vulnerability or diathesis-stress models (diathesis being essentially a synonym for vulnerability).

1.1.1 Zubin and Spring's Vulnerability Model

The vulnerability model of schizophrenia articulated by Zubin and Spring (1977) was a landmark in the evolution of modern systemic models. In addition to its conceptual innovation, the Zubin and Spring (1977) model gave a much improved account of the disparate empirical evidence from rigorous research. The key concept in the model was a distinction between vulnerability to schizophrenia – a relatively stable, long-lasting trait – and the instable, oscillating states (acute psychotic episodes) recognized as diagnostic features of schizophrenia (see Figure 1.1).

The original Zubin and Spring (1977) vulnerability model was revised and elaborated by various theorists over the following years. However, the key concept remained: pre-

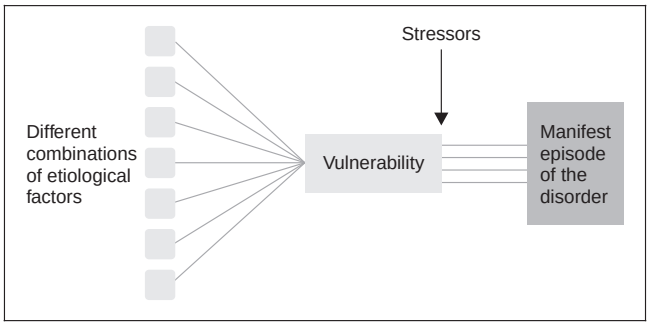

Figure 1.1: Zubin and Spring's vulnerability model (1977) as depicted by Brenner (1989)

morbid vulnerability associated with various causal factors. According to vulnerability models, a diagnosable episode of schizophrenia occurs only if a vulnerable individual is subjected to certain stressful demands. In individuals with more pronounced levels of vulnerability, minor demands can trigger an episode, whereas more severe stress may be required to trigger an episode in individuals with more moderate levels of vulnerability.

1.1.2 Nuechterlein and Colleagues' Heuristic Vulnerability/Stress Model

A more detailed and complex vulnerability model for schizophrenia was assembled by Nuechterlein and his colleagues (Nuechterlein & Dawson, 1984a; Nuechterlein, Dawson, & Green, 1994; see Figure 1.2). It was termed a *heuristic model* because its creators recognized that it identified only a subset of all possible vulnerabilities and causal pathways, but it did provide a reasonably complete description of the ways in which vulnerability factors might interact with other factors to produce the diagnosed disorder. In this model, a relatively stable vulnerable state is produced by biological factors, most importantly a dysfunction of dopamine neurotransmitter systems. Other features of the vulnerable state may include an instability and a hyperreactivity of the autonomic nervous system, cognitive deficits, and maladaptive social/behavioral traits. There may also be protective factors that may, at least partially, compensate for impairments caused by the vulnerabilities and/or mitigate the effects of stress, examples being high self-efficacy, good problem-solving skills, and a supportive family. Antipsychotic medication is a kind of protective factor that buffers a vulnerable central nervous system against neurophysiological dysregulation. The expression of diagnosable schizophrenia occurs when the net influence of all vulnerabilities overcomes the contributions of protective factors. Finally, the model of Nuechterlein and colleagues (1994) includes impairments of cognitive and autonomic functioning, which intermediate between stability and instability. These are the mechanisms by which the diagnosed disorder is expressed.

1.1.3 Elaborations of Systemic Vulnerability Models

Systemic vulnerability models of schizophrenia inspired the search for specific expressions in biological, psychological, and behavioral levels of functioning. A number of

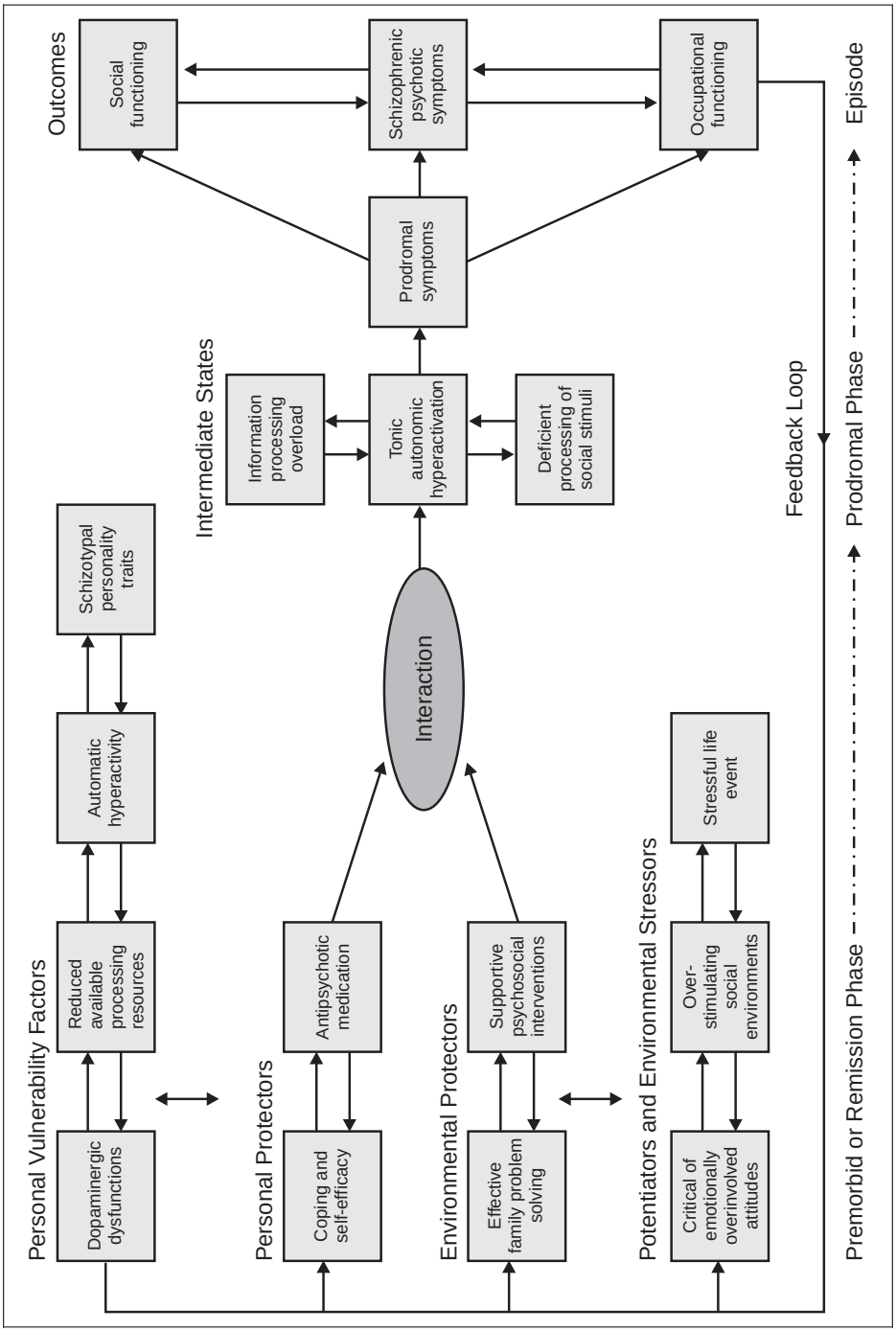

Figure 1.2: Heuristic vulnerability/stress model (Nuechterlein & Dawson, 1984a,b; Nuechterlein, Dawson, & Green, 1994)

such expressions were found, and several of them are described in this section. In some cases, these expressions may play a mechanistic role in etiology, such as causal links between a vulnerability genotype and a functional brain impairment. These mediating factors are receiving increasing attention in research on the endophenotypes of vulnerability. In other cases, the expressions may be measurable indications of vulnerability, but may play no role in the actual etiology of the disorder. These are generally termed *vulnerability markers*.

Genetic Factors

Genetic factors are still thought to be the primary source of biological vulnerabilities to schizophrenia. The concordance rate for monozygotic twins is estimated to be 50%–70% (Gottesman & Shields, 1982; Kendler et al., 1993; Sullivan, Kendler, & Neale, 2003). The remaining 30%–50% corroborate the vulnerability hypothesis that genetic factors interact with environmental factors to produce the disease (Mueser & McGurk, 2004; Portin & Alanen, 1997a,b; Tandon, Keshavan, & Nasrallah, 2008). A large number of specific genes are probably involved in vulnerability, and the specific mechanisms may range from structural flaws in dopamine system receptors to an immunological vulnerability to viral infection during gestation (van Os, Krabbendam, Myin-Germeys, & Delespaul, 2005).

Neurotransmitter Regulation

Dopamine remains a key neurophysiological factor in systemic models of schizophrenia, especially in the mesolimbic dopamine system (e.g., Broome et al., 2005; Kapur, Mizrahi, & Li, 2005). All antipsychotic medications act on the dopamine system, in particular on the D2 receptor (Garety, Bebbington, Fowler, Freeman, & Kuipers, 2007). Dopamine agonists, such as amphetamine, can precipitate psychosis in vulnerable individuals (Howes, Asselin, Murray, McGuire, & Grasby, 2006). Earlier hypotheses of an overabundance of dopamine have given way to hypotheses about the distribution and sensitivity of dopamine receptors. However, alternative models abound and the precise role of dopamine regulation as either a vulnerability marker or an endophenotype of schizophrenia is still not well understood. Generalized dopamine dysregulation clearly plays a key role in acute psychosis, and specific abnormalities in dopamine mechanisms may create a vulnerability to generalized dopamine dysregulation.

Brain Structure and Function

New medical imaging technologies, such as computer assisted tomography (CAT), positron emission tomography (PET), and magnetic resonance imaging (MRI), have stimulated much research on brain structure and function in schizophrenia (e.g., Blakemore & Frith, 2000; Buchanan & Carpenter, 1997; Kile, 2007). Initial studies failed to reveal differences in the brains of schizophrenia patients and normal controls, although this

changed as imaging technology improved (Chua & McKenna, 1995). One of the most replicated findings is an enlargement of the lateral ventricles in schizophrenia patients, compared to normal controls (Crespo-Facorro et al., 2007; Kile, 2007; Lombardo-Ferrari, Kimura, Nita, & Elkis, 2006; Ohara, Sato, Tanabu, Yoshida, & Shibuya, 2006; Raz & Raz, 1990; Sharma et al., 1998; Van Horn & McManus, 1992). Nevertheless, ventricle enlargement is neither a necessary nor a sufficient factor in the development of schizophrenia (Stevens, 1997). Studies of identical twins discordant for schizophrenia suggest that ventricular enlargement is rather an indirect indication of subtle anatomical abnormalities in the limbic system (Suddath, Christison, Torrey, Casanova, & Weinberger, 1990), possibly due to developmental neurodysplasia.

Dynamic imaging, such as PET and functional MRI, tends to indicate reduced metabolic activity in the frontal lobes of schizophrenia patients compared to controls. This pattern is generally termed *hypofrontality* (for reviews see Blakemore & Frith, 2000; Ragland, Minzenberg, & Carter, 2007). Metabolic hypofrontality appears to be associated with impairments in neuropsychological functions in the frontal cortex, for example, working memory and executive functioning (Fu et al., 2005; MacDonald et al., 2005). This could form a link between the biological and cognitive levels of a specific type of vulnerability.

Specific brain structures implicated in schizophrenia include the dorsolateral, prefrontal, and temporal cortex, the various limbic system structures, the basal ganglia, parts of the thalamus, and the cerebellum (Andreasen, Paradiso, & O'Leary, 1998; Camchong, Dyckman, Chapman, Yanasak, & McDowell, 2006; Lee et al., 2006; Rüsch et al., 2007). The diversity of findings has inspired a more integrative perspective on brain impairments. For example, the cortical dysconnectivity syndrome model hypothesizes that normal interaction between multiple brain areas is impaired by synaptic processes common to those areas (James, James, Smith, & Javaloyes, 2004; Vogeley & Falkai, 1998; Wobrock et al., 2008). The failure is not situated in a specific brain structure or area, but in the transmission of information between them. Similarly, there is empirical evidence for fronto-temporal dissociation, or impaired transmission between frontal and temporal areas, in schizophrenia patients (Murray et al., 2008; Woodruff et al., 1997).

Cognition

Cognitive abnormalities have long been seen as core features of schizophrenia, arguably since Kraepelin (Green, 1998). Systemic vulnerability models stimulated new hypotheses about the etiological roles of these abnormalities – as markers, endophenotypes, and expressions of the disease. Soon after the first vulnerability models had appeared, cognitive abnormalities were found in children at risk (e.g., Asarnow, Steffy, MacCrimmon, & Cleghorn, 1978; Erlenmeyer-Kimling & Cornblatt, 1978) and in unaffected first-degree relatives (DeAmicis & Cromwell, 1979; Sitskoorn, Aleman, Ebich, Appels, & Kahn, 2004). In longitudinal studies, in support of the vulnerability hypothesis, impaired attention, and related information processing in children at risk for schizophrenia is related to eventual onset (Erlenmeyer-Kimling et al., 2000). Such

abnormalities are also associated with impairments in social functioning in adulthood (Cornblatt, Lenzenweger, Dworkin, & Erlenmeyer-Kimling, 1992), suggesting more than a mere marker role in etiology.

As the cognitive research progressed, it became important to distinguish between trait-like and state-like abnormalities (Nuechterlein & Dawson, 1984b; Nuechterlein & Subotnik, 1998; Özgürdal et al., 2009; Wang, Chan, Yu, Shi, & Deng, 2008; Wykes & van der Gaag, 2001). Vulnerability factors tend to be more trait-like, present before as well as after onset, and relatively stable in severity (e.g., Aleman, Hijman, deHaan, & Kahn, 1999; Kurtz, 2005; Woods, Twamley, Dawson, Narvaez, & Jeste, 2007); state-like abnormalities tend to be associated with the acute psychotic state (e.g., Baxter & Liddle, 1998; Filbey et al., 2008; Nopoulos, Flashman, Flaum, Arndt, & Andreasen, 1994). Some abnormalities show characteristics of both, present at low levels before onset and between episodes, and becoming more severe after onset and/or during acute psychotic episodes. Many cognitive impairments appear to exert their effects over the course of the illness, producing poorer treatment adherence (Jeste, Patterson, Palmer, Dolder, & Jeste, 2003), functional behavioral impairments (Frith, 1992), higher risk of relapse (Chen et al., 2005), and a poorer overall prognosis (Green, Kern, Braf, & Mintz, 2000; McEvoy, 2008; Milev, Ho, Arndt, & Andreasen, 2005).

Another distinction of increasing importance is that between *neurocognition* and *social cognition* (Corrigan & Penn, 2001; Green, Penn et al., 2008; Penn, Corrigan, Bentall, & Racenstein, 1997; Penn et al., 2005). Most of the cognitive processes of interest in vulnerability research are relatively elemental, for example, reaction time, attention, and memory, derived from the laboratory methods of experimental psychopathology and clinical neuropsychology. These cognitive processes were used in a non-social context as neurobiological correlates (neurocognition). More recently, however, more complex levels of cognition have attracted the interest of schizophrenia researchers. The terms neurocognition and social cognition came to distinguish between these subdomains, although they are not necessarily distinct or nonoverlapping. Social cognition refers to how people think about themselves and others in the social world; simply put, it is people's thinking about people (Green, Olivier, Crawley, Penn, & Silverstein, 2005; Penn, Sanna, & Roberts, 2008). Interest in social cognition was inspired in part by the fact that, although more elemental measures appear linked to vulnerability factors, their actual mechanistic role in etiology has been difficult to establish. Green and Nuechterlein (1999) proposed a model including social cognition as a possible mediator between neurocognitive and behavioral functioning, and empirical evidence supports such a role (Addington, Saeedi, & Addington, 2006b; Bell, Tsang, Greig, & Bryson, 2008; Brekke & Nakagami, 2010; Brekke, Kay, Lee, & Green, 2005; Brüne, 2005; Pinkham & Penn, 2006; Pinkham, Penn, Perkins, & Lieberman, 2003; Roder & Schmidt, 2009; Schmidt, Mueller, & Roder, 2009; Sergi, Green et al., 2007; Sergi, Rassovsky, Nuechterlein, & Green, 2006; Sergi, Rassovsky et al., 2007; Vauth, Rüsch, Wirtz, & Corrigan, 2004). Also, since social cognition is more functionally proximal to the deficits in social behavior that are of primary clinical concern in schizophrenia, it is hoped that a better understanding of social cognition will lead more directly to improved clinical assessment and treatment methods (Addington et al., 2006b).

Specific social cognitive impairments under current study include theory of mind (a person's ability to infer the cognitive and emotional states of other people), social schema, social attribution, and social perception (Bellack, Morrison, & Mueser, 1989; Corcoran, Mercer, & Frith, 1995; Frith, 2004; Nienow, Docherty, Cohen, & Dinzeo, 2006; Penn et al., 1999; Pinkham, Penn, Perkins, Graham, & Siegel, 2007; Toomey, Wallace, Corrigan, Schuldberg, & Green, 1997; Zanello, Perrig, & Huguelet, 2006). Our understanding of this level of organismic functioning will probably expand considerably over the next few years. Many aspects of social cognition pertinent to severe mental illness have likely not yet been identified or measured. Also, the relationship between social cognition and neurocognition may not be purely hierarchical: There are differences between processing of social versus nonsocial information at fairly elemental levels, for example, between visual processing of the physical features of alphanumeric characters versus human faces.

Environmental Factors

Studies of the stress side of the vulnerability hypothesis were influenced by two distinct research paradigms: life events and expressed emotion. Life-events research typically involves an enumeration of specific stressful events that may either accumulate over time to interact with vulnerabilities or precipitate onset in a single stroke (Bebbington et al., 1993; Day, 1989; Dohrenwend, Shrout, Link, Skodol, & Stueve, 1995; Phillips, Francey, Edwards, & McMurray, 2007; Tennant, 1985). Both types of mechanisms appear to operate in the etiology of schizophrenia. After onset, both major stressors and minor "daily hassles" may influence the course of the disorder. Recently, the disproportionate representation of persons with histories of major trauma, childhood abuse, and childhood neglect among those with serious mental illness has given life-events research a new focus (Bebbington et al., 2004; Janssen et al., 2004; Read, van Os, Morrison, & Ross, 2005; Spauwen, Krabbendam, Lieb, Wittchen, & van Os, 2006). As with other life events, trauma appears to influence the course of the disorder over time as well as to precipitate the onset (Schenkel, Spaulding, DiLillo, & Silverstein, 2005).

Expressed emotion research has evolved from early findings that the emotional climate in a family has an impact on the stability of family members with schizophrenia (Leff & Vaughn, 1985). Stress in a high-expressed emotion family may be produced by family members' negative, critical attitudes – or by well-intended overinvolvement with the identified patient (Brown, Birley, & Wing, 1972; Butzlaff & Hooley, 1998; Hooley, 2007). Therapeutic techniques designed to reduce the expressed emotion in family networks demonstrated clear clinical efficacy in preventing relapse (Pilling et al., 2002).

Environmental stress relevant to vulnerability for schizophrenia may come from a variety of other sources. Stressors known to influence the incidence of schizophrenia include urban birth (McGrath, 2006), lack of social cohesion (Kirkbride et al., 2007), emigration (Smith et al., 2006), poverty (Cohen, 1993), and war (van Os & Selten, 1998). A model of vulnerability to schizophrenia (Walker & Diforio, 1997)

proposes links between extreme environmental stress, fetal exposure to maternal cortisol (a hormone secreted in response to stress), developmental neurodysplasia, and vulnerability.

1.1.4 Pervasiveness and Homeorhesis

Another landmark in the evolution of systemic vulnerability models of schizophrenia was the concept of *pervasiveness* (Brenner, 1986; Spaulding, 1986). Inspired by vulnerability models, experimental psychopathology had produced a proliferation of laboratory paradigms that revealed abnormalities associated with schizophrenia (Cromwell & Spaulding, 1978). The processes measured by these paradigms were mostly cognitive, but they spanned a range of complexity, from elemental visual feature processing, to attention, to trait-like characteristics of social judgment and attribution. Borrowing from concepts originating in learning theory, these processes were ordered on a continuum ranging from relatively molecular to relatively molar levels of organization. Consistent with the information-processing models of cognition that predominated at that time, it was hypothesized that information is passed from more molecular processes to more molar processes in the course of apprehending the environment and responding to it. In the context of psychopathology, this means that impaired molecular processes pass corrupted information to more molar processes, thus distributing the effects upward throughout the cognitive system. Similarly, failures in the cognitive level of organismic functioning would be distributed to the behavioral and social levels. Schizophrenia came to be understood as a pervasive disorder, with significant impairments at every level of biological, psychological, behavioral, and social functioning.

As systemic theories became increasingly influential in psychology (e.g., Bronfenbrenner, 1979), the pervasiveness concept in schizophrenia research continued to evolve. Linear models of information processing, in which deficits are passed upward from molecular to molar levels, gave way to nonlinear models, wherein deficits are passed in both directions, within and between levels of functioning. The effects of vulnerabilities are reciprocal. For example, impaired attention can induce paranoid interpretations of social behavior, which in turn can compromise performance of social skills. Deficient social skills put greater demands on attention and create stress, which has neurophysiological consequences. The neurophysiological consequences of stress exacerbate attention deficits and paranoia, further impairing social skills. Impaired social skills also create a more stressful social environment, as family members struggle to respond. The functional deficits of schizophrenia came to be seen as the results of vicious circles between specific impairments (Brenner, Hodel, Roder, & Corrigan, 1992).

This insight into the reciprocal interactions of specific impairments or vulnerabilities intersected with the evolution of cognitive perspectives in psychology and psychopathology (as discussed in the previous section). The paradigms that had dominated experimental psychopathology and neuropsychology were under increasing attack as paradigms of "cold" cognition, meaning paradigms that analyze cognition in contrived laboratory conditions. The new concept of social cognition was developed in part to study "hot" cognition, meaning cognition with personal relevance and emotional sig-

nificance as it actually occurs in natural settings. Systemic models that posit reciprocal interactions between cognition and other levels of organismic functioning and environmental events are better suited to understanding the role of "hot" social cognition in psychopathology (Penn et al., 2008). Integration of personally relevant material in laboratory assessments of social cognition is not yet common, but this may become an important development in the near future.

Systemic psychological models have implications for human development, and this applies to psychopathology as well. The familiar concept of homeostasis, referring to the static stability of a biological system, is now complemented by the concept of *homeorhesis*. A system in homeorhesis is homeostatic, in the sense that it is stable in the short term, but in the long term undergoes gradual changes. Humans are in homeorhesis because, while they maintain short-term homeostasis, they grow and change in various ways throughout the life course. Sometimes the change is desirable – as in the maturation of children – and sometimes less desirable, as in advanced aging. Eventually a deteriorating homeorhetic system may reach a point of collapse, in which homeostasis is disrupted, such as in death.

Schizophrenia can be understood as a condition of homeorhesis. In the short term, people with schizophrenia sustain a degree of homeostasis, preserving basic functioning despite some number of vulnerabilities that compromise the efficiency and long-term stability of their organismic system. However, the inefficiencies and the environmental stress they cause create a long-term tendency toward less efficiency and more stress. When extended over time, vicious circles become downward spirals, and at some point the spiral may accelerate enough to create a cascading system collapse, resulting not in death but in acute psychosis. Thus, advanced system theories and the concept of homeorhesis provide an expanded understanding of the actions of vulnerabilities over time.

The developmental dimension of system theory also sheds new light on how impairments become pervasive. An impaired component influences the rest of the system, not just at one point in time, but over time as human development progresses. For example, the cumulative effect of an attention deficit on social skills is not just during the moment of skill performance, but over the developmental period during which social skills are being acquired and refined. This insight was particularly important in light of the finding that crucial aspects of neuropsychological development extend through adolescence and early adulthood (e.g., Kolb & Nonneman, 1976). Since the onset of schizophrenia typically occurs in late adolescence or early adulthood, it disrupts some crucial developmental processes, leaving the person without specific cognitive abilities that normal adults take for granted.

The implications of advanced system theory define and guide an era of schizophrenia research that continues to this day. The implication that functional deficits are caused by impairments distributed throughout the system stimulated analysis across multiple levels of organismic functioning (e.g., Addington & Addington, 1999; Brekke et al., 2005; Green, 1996; McGurk & Mueser, 2004; Prouteau et al., 2005; Semkovska, Bedard, Godbout, Limoge, & Stip, 2004; Wykes & van der Gaag, 2001). These analyses provide empirical support for the systemic nature of schizophrenia.

1.2 Systemic Models in Clinical Application

One particularly important implication of biosystemic models is that treatment of schizophrenia should address multiple levels of functioning in a coordinated way. This was the key idea behind the development of Integrated Psychological Therapy (IPT). In the broadest sense it is also one of the key principles of psychiatric rehabilitation as such (Liberman, 2008; Spaulding, Sullivan, & Poland, 2003), and generally regarded as the comprehensive treatment approach of choice for severe mental illness, an approach in which modalities like IPT form core components. Outcome research on the effectiveness of treatments that address multiple levels of functioning provide empirical support for the hypothesis that a systemic approach to treatment meaningfully enhances recovery from schizophrenia (e.g., Cohen, Forbes, Mann, & Blanchard, 2006; Dickerson, Boronow, Ringel, & Parente, 1999; Hofer et al., 2005; Liddle, 2000; Milev et al., 2005; Reeder, Smedley, Butt, Bogner, & Wykes, 2006; Revheim et al., 2006; Roder, Mueller, Mueser, & Brenner, 2006; Spaulding, Reed, Sullivan, Richardson, & Weiler, 1999; Twamley, Savla, Zurhellen, & Heaton, 2008; Twamley, Woods et al., 2008; Velligan et al., 2000; Wykes, Reeder, Corner, Williams, & Eyeritt, 1999).

The remainder of this chapter discusses some key considerations in comprehensive application of biosystemic models in clinical assessment, treatment, and rehabilitation.

1.2.1 Cognitive Science and Technology

As discussed previously in this chapter, cognitive research has had a significant impact on the evolution of systemic vulnerability-stress models of schizophrenia. Accordingly, there has been a growing awareness of the need for new cognitive technology for clinical assessment and treatment (Spaulding, 1994).

A key empirical finding of the past 20 years of schizophrenia research was the ubiquity of neurocognitive impairments in people with schizophrenia. As many as 75%–85% of all schizophrenia patients have persistent deficits on neuropsychological performance measures, independent of patients' age (Bowie, Reichenberg, McClure, Leung, & Harvey, 2008; Gray & Roth, 2007). The average patient scores two standard deviations below healthy controls on neuropsychological tests, lying in the lowest 5%–10% of the general population (Keefe, 2007; Wilk et al., 2004). These findings corroborate a traditional view of schizophrenia, arguably dating back to Kraepelin (Green, 1997) but certainly to Bleuler (1911), namely, that cognitive impairments are distinctive and even defining features of the illness. In modern terminology, schizophrenia can be considered a *neurocognitive disorder* (Green, 1998). As discussed earlier in this chapter, social cognitive paradigms also promise to add much to our understanding of the cognitive impairments of schizophrenia (e.g., Bigelow et al., 2006; Horan et al., 2009; Kee et al., 2009; Sprong, Schothorst, Vos, Hox, & van Engeland, 2007).

Despite the prevalence and severity of cognitive impairment in schizophrenia, there is no single type or profile that characterizes the illness. Heterogeneity, in both the

quality and severity of impairments, is the rule. A substantial minority of schizophrenia patients score in or above the average range on neuropsychological tests. There is sufficient variability that cognitive measures can predict within-group differences on other dimensions, for instance, treatment compliance and risk of relapse in first-episode patients (Chen et al., 2005) and longer-term outcome in more chronic patients (e.g., Brekke et al., 2005; Norman et al., 1999; Peer & Spaulding, 2007; Silverstein, Harrow, & Bryson, 1994; Silverstein, Mavrolefteros, & Close, 2002; Straube, 1993). This means that, for clinical purposes, assessment must be able to identify and characterize individual cognitive differences between people with schizophrenia – and treatment must be flexible enough to accommodate these differences.

The increasing recognition of the importance of cognitive factors in schizophrenia produced a proliferation of treatment approaches and assessment tools. Integrated Psychological Therapy (IPT) is representative of an array of treatment modalities that target neurocognitive and social cognitive impairments. The proliferation of assessment tools has, in fact, been so great that there are too many to ensure any comparability of findings across research studies. Concern about this has become great enough that a major effort to standardize cognitive assessment in schizophrenia research was undertaken by the U. S. National Institute of Mental Health (NIMH), the Measurement and Treatment Research to Improve Cognition in Schizophrenia (MATRICS). This initiative reflects widespread recognition (1) that cognitive factors are central to the understanding and treatment of schizophrenia, but (2) that the complexity of cognition and a proliferation of assessment tools threaten to inhibit scientific progress by generating large amounts of data that cannot be compared across studies or research groups. The solution to the problem was to assemble a single battery of cognitive measures, broad enough in scope to cover the major domains of cognition implicated in schizophrenia but small enough to be highly portable and suitable for a wide range of research applications (Green & Nuechterlein, 2004; Kern & Horan, 2010; Nuechterlein et al., 2004). A major motivation in the MATRICS initiative was to produce a battery well suited for research on psychopharmacological treatment of cognitive impairment. However, it was also expected that a standardized battery suitable for that purpose would also be suitable for a broad range of applications.

The MATRICS initiative involved a large number of schizophrenia researchers in a process of reaching consensus about the optimal contents of a cognitive assessment battery for schizophrenia research. The initial focus was on the neurocognitive domain, because the proliferation of assessment tools had been greatest in that domain, and there was a great desire to find psychopharmacological agents with benefits in that domain. After considerable deliberation, six independent subdomains of neurocognition relevant for schizophrenia disorders were identified on the basis of assessments used in research (Nuechterlein et al., 2004):

- **Speed of information processing:** This domain emphasizes the speed of performance including perceptual and motor components with which information is processed (e.g., cognitive flexibility, sensomotor speed).
- **Attention/Vigilance:** Selective attention works as a filter, with the function of selecting information prior to its further treatment according to its importance. Insufficient filtering of the mass of information and a lack of inhibition of irrelevant stim-